Amendments to the Specification:

Please replace the OTHER PUBLICATIONS section beginning at page 3, line 4 with the following amended section:

~~OTHER PUBLICATIONS~~ NON-PATENT DOCUMENTS

[1] Wang et al, ~~H.H. & Wang, X.F.~~ In Progress in atherosclerosis research: Analytical methods for atherosclerosis research. ~~(ed. by Columbus F.) In press (~~ Editor Schoenhagen, Nova Science Publishers Inc., ~~New York, 2004).~~ 2006, PP.33-66.

[2] Wang, [[H.H.]] Analytical models of atherosclerosis. Review. Atherosclerosis, ~~159, 1-7 (2001).~~ 2001, Vol.159, PP.1-7.

[3] Grundy, [[S.C.]] In Plasma lipoproteins and coronary artery disease: Role of low-density lipoproteins in development of coronary artery atherosclerosis. ~~(eds. by Kreisberg, R.A. & Segrest, J.P.) 93-124 (Blackwell Scientific, Cambridge, 1992).~~ Editor Kreisberg et al., Blackwell Scientific, 1992, PP.93-124.

[4] National Cholesterol Education Program. Second report of the expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult treatment panel II). Circulation, ~~106 (25), 1333-1445 (2002).~~ 2002,

Vol.106, PP.1333-1445.

[5] Shephered et al, ~~J., Betteridge, D.J. & Durrington, P.~~ Strategies for reducing coronary heart disease and desirable limits for blood lipid concentrations: guidelines from the British Hyperlipidaemia Association. ~~Br. Med. J. 295, 1245-1246 (1987).~~ British Medicine Journal, 1987, Vol.295, PP.1245-1246.

[6] Study group of the European Atherosclerosis Society. The recognition and management of hyperlipidaemia in adults. A policy statement of European Atherosclerosis Society. ~~Eur. Heart. J. 9, 571-600 (1988).~~ Europe Heart Journal, 1988, Vol.9, pp.571-600.

[7] Canadian lipoprotein conference at hoc committee on guidelines for dyslipoproteinemias. Guidelines for the detection of high risk lipoprotein profiles and the treatment of dyslipoproteinemias. ~~Can. Med. Assoc. J. 142, 1371-1382 (1990).~~ Canada Medicine Association Journal, 1990, Vol.142, pp.1371-1382.

[8] National Center for Health Statistics. National health and nutritional examination, ~~survey (III), (1994).~~ 1994, Survey (III).

[9] Libby, [[P.]] Inflammation in atherosclerosis. Review. Nature, ~~420, 868-874 (2002).~~ 2002, Vol.420, pp.868-874.

[10] Li et al, ~~A.C. & Glass, C.K.~~ The macrophage foam cell as a target for therapeutic intervention. Review. Nature Medicine, ~~8, 1235-1242 (2002).~~ 2002, Vol.8, pp.1235-1242.

[11] Ross et al, ~~R. & Glomset, J. Atherosclerosis and arterial smooth muscle cell. Science 180, 1332-1339 (1973). Science, 1973,~~ Mechanisms of disease: Atherosclerosis-an inflammatory disease, New England Journal Medicine, 1999, Vol.340, pp.115-126.

[12] Caro et al, ~~C.G., Fitzgerald, J.M. & Schroter, R.C.~~ Arterial wall shear and distribution of early atheroma in man. Nature, ~~223, 1159-1161 (1969).~~ 1969, Vol.223, pp.1159-1161.

[13] Texon,[[M.]] Hemodynamic basis of atherosclerosis. [[()]]Hemisphere Publishing Corporation, ~~Washington,~~ 1980[[)]].

[14] Friedman[[, M.H.]] et al, ~~Deters, O.J., Mark, F.F., Bargeron, C.B. & Hutchins, G.M.~~ Arterial geometry affects hemodynamics: a potential risk factor for atherosclerosis. Atherosclerosis, ~~46, 225-231 (1983).~~ 1983, Vol.46, pp.225-231.

[15] Beere et al, ~~P.A., Glagov, S. & Zarins, C.K.~~ Retarding effect of lowered heart rate on coronary atherosclerosis. Science, ~~226, 180-182 (1984).~~ 1984, Vol.226, pp.180-182.

[16] Kannel et al, ~~W.B., Kannel, C. & Paffenbarger,~~

~~R.S.J.~~ Heart rate and cardiovascular mortality: The Framingham study. ~~Am. Heart. J. 113, 1489-1494 (1987).~~ America Heart Journal, 1987, Vol.113, pp.1489-1494.

[17] Schwartz et al, ~~C.J., Valente, A.J., Sprague, E.A., Kelley, J.L. & Nerem, R.M.~~ The pathogenesis of atherosclerosis: an overview. ~~Clin. Cardiol. 14, 1-16 (1991).~~ Clinical Cardiology, 1991, Vol.14, pp.1-16.

[18] Kruth, [[H.S.]] Lipoprotein cholesterol and atherosclerosis. Review. Current Molecular Medicine, ~~1, 633-653 (2001).~~ 2001, Vol.1, pp.633-653.

[19] Lusis, [[A.]] Atherosclerosis. Review. Nature, ~~407, 233-241 (2000).~~ 2000, Vol.407, pp.233-241.

[20] Could[[, A.L.,]] et al., Cholesterol reduction yields clinical benefit: Impact of statin trials. Circulation, ~~97 (10), 946-952 (1998).~~ 1998, Vol.97, pp.946-952.

[21] Debakey et al, ~~M.E., Lawrie, G.M. & Glaeser, D.H.~~ Patterns of atherosclerosis and their surgical significance. ~~Ann. Surge. 201, 115-131 (1985).~~ Annual Surgery, 1985, Vol.201, pp.115-131.

[22] Bargeron et al, ~~C.B., Hutchins, G.M. & Moore, G.W.~~ Distribution of the geometric parameters of human aortic bifurcations. Atherosclerosis, ~~6, 109-113 (1986).~~ 1986, Vol.6, pp.109-113.

[23] Ravensbergen et al., The influence of the angle of confluence on the flow in a vertebro-basilar junction model, Journal of Biomechanics 1996, Vol.29, No.3, pp.281-299.

[24] Ballantyne et al., Role of lipid and lipoprotein profiles in risk assessment and therapy, The American Heart Journal, 2003, August; Vol.146, No.2, Abstract.

[25] Evans et al., Medical lipid-regulating therapy: Current evidence, ongoing trials and future developments, Drugs, 2004, Vol.64, No.11, Abstract.

Please replace paragraph [0012] with the following amended paragraph:

[0012] The present invention is a multiparameter screening method that is used for combining the contributions of atherosclerotic risk factors to the disease, predicting a total risk of the disease and a disease risk level, determining a primary cause in the disease, assessing a therapeutic efficacy and optimizing the therapeutic targets at the different stages of the disease in different individuals who require the diagnosis, prevention or treatment of atherosclerosis-related CHD or stroke, which comprises the following phases:

defining the normal as free from atherosclerosis-

related coronary heart disease or stroke;

the measured values refer to the quantities of atherosclerotic parameters to be measured;

an individual having the measured values of atherosclerotic parameters;

~~determining~~ the individual having the normal values of these atherosclerotic parameters;

determining the disease risks yielded by the differences between the measured values and the normal values of these atherosclerotic parameters;

adding all the disease risks together so as to yield a total risk of the disease;

determining a disease risk level containing the total risk of the disease;

selecting an atherosclerotic risk factor related to an atherosclerotic parameter that is the greatest contribution to the total risk so as to result in this risk factor as a primary therapy target of the disease;

determining a greater flux between the LDL mass transfer flux and the monocyte mass transfer flux so as to result in this greater flux as a primary cause in the disease;

selecting a greater concentration level between the LDL level in serum and the CRP level in blood plasma so as to result in this greater level as a secondary therapy target of the disease;

calculating a relative ratio between the current total risk from the currently measured values of these atherosclerotic parameters and the previous total risk from previously measured values of these parameters so as to yield this ratio as a therapeutic efficacy of the disease; and

repeating the above-mentioned methods until the disease risk level is reduced to a normal level for the individual who requires the therapy to prevent or to treat atherosclerosis-related CHD or stroke.

the above-mentioned methods are written as an executable computer program named the MMA.exe to ~~perform~~ be installed into a general purpose digital computer device to accomplish said methods.

Please replace paragraph [0025] with the following amended paragraph:

[0025] Substituting (C), (E) and (F) into (1.1) yields

$$J = Bc^{\frac{11}{9}} p^{\frac{1}{3}} T^{\frac{16}{27}} a^{\frac{2}{3}} f^{\frac{2}{9}} z^{-\frac{2}{9}} \qquad (1.2)$$

and

$$J = Ec^{\frac{11}{9}} D^{\frac{16}{27}} z^{-\frac{2}{9}} (\cos\alpha)^{\frac{2}{9}} \qquad (1.3)$$

where J = the mass transfer flux in 10^{-5} g/cm^{2}s; the

atherosclerotic parameters including c = the LDL concentration parameter in mg/dL or c = the CRP concentration parameter in mg/L, p = the blood systolic pressure parameter in mmHg or p = the blood diastolic pressure parameter in mmHg, f = the heart rate parameter in s^{-1}, T = the plasma temperature parameter in °C, α = the angle parameter in degree, a = the radius parameter of arterial vessels in cm, and z = the axial position parameter of diffusional flux in cm or z is called the diffusional length; D = the diffusion coefficient in cm^2/s; the ~~variable~~ conversion factor $B = AH_a^{\frac{1}{3}} H_b^{\frac{1}{9}} H_d^{\frac{16}{27}}$ that is independent of c, p, T, f, a and z in (1.2); and the ~~variable~~ conversion factor $E = Ag\nu^{\frac{3}{27}}$ that is independent of c, D, α and z in (1.3).

Please replace paragraph [0048] with the following amended paragraph:

[0048] Step eleven: These methods in Step three through Step nine are written as an executable computer program named said MMA.exe ~~that provides greater ease and convenience to perform~~ to be installed into a general purpose digital computer device to accomplish these methods comprising:

starting the MMA.exe program on the device;

inputting the currently measured values, the previously measured values and the normal values of the individual's atherosclerosis parameters into the MMA.exe input screen by using the keyboard of the device;

clicking the "update" button and the "calc. risk" button of the input screen by using the mouse of the device; and

clicking the "evaluate" button of the output screen so as to yield the screening results including a total risk of the disease, a primary cause in the disease, a primary therapy target of the disease, a secondary therapy target of the disease and a therapeutic efficiency for individuals who require the therapy to prevent or treat atherosclerosis-related CHD or stroke.

Please replace paragraph [0059] with the following amended paragraph:

[0059] Example 4. Autopsy and clinical studies [13-14, 17, 21] suggested that regions of arterial bifurcations had the greatest predilection for atherosclerosis. However, [[no]]the current screening method such as screening LDL or cholesterol levels in the patients' blood ~~is able~~ is unable to determine

the contribution of the arterial geometry to the disease. Internal angles among 70 human aortic bifurcations can vary widely from 10° to 70° [22]. Different internal angles may lead to different angle α in (1.3).